AGEING BETWEEN GERONTOLOGY AND BIOMEDICINE

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Abstract: Over the past two decades, scientific, public and economic interest in the basic biological processes underlying the phenomenon of ageing has grown considerably. New developments in biotechnology and health maintenance programmes appear to be forging new relationships between biology, medicine and the 'ageing society'. Social scientific research on these changes has captured the process under the concept of 'biomedicalisation of aging', through which ageing becomes increasingly understood and managed as a biological problem. In this paper, we argue instead that contemporary biogerontology is at odds with what has come to be known as biomedicine in the second half of the 20th century and that it can be said to represent a critique of biomedicine. We propose a genealogy of this critique and argue that it is shaped by two interrelated and historically contingent conditions, namely the status of biogerontologists as 'outsiders' to the institutions of biomedicine, and, more importantly, the hybrid character of biomedicine itself. Finally, we point however towards sources of uncertainty within biogerontogy that should be taken into account in further research.

KEYWORDS: Biogerontology; biomedicine; biomedicalisation of ageing; evolutionary medicine; health and ageing; governance of ageing.

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Introduction

Over the past two decades, interest in the basic biological processes underlying the phenomenon of 'ageing' has grown considerably. The emergence of the so-called 'anti-ageing' movement and the controversies surrounding its promises have certainly captured the attention of British and American publics, but so have the more sober assessments presented in popular books such as Leonard Hayflick's *How and Why We Age* (1994) and Thomas Kirkwood's *The End of Age: The Science of Human Aging* (1999). This interest has motivated both consultations on public attitudes toward research into the causes of ageing (Alliance for Aging Research, 2005; MORI, 2006) and political interventions such as the White House Conference on Aging (2005) and the House of Lords Report on Ageing (2005). These developments have been accompanied by an intensification of research on the biology of ageing, and a number of bio-molecules have been characterised that are currently being tested in animal and human trials aiming to assess their therapeutic value. Lastly, clinicians would appear to be willing increasingly to provide these and other life-extending treatments to older individuals (Kaufman et al., 2004). Not surprisingly, this entire situation has captured the attention of social scientists, many of whom refer to the 'biomedicalization of aging'. Estes and Binney (1989) first coined the phrase 'biomedicalization of aging' as a conceptual label for processes whereby ageing comes to be defined as a matter of 'biomedical' interest, processes which would appear today to be intensifying and increasingly associated with the reorganisation of health care around technological intervention and the modes of prevention and consumption (Clarke et al., 2003; Rose, 2007). Contemporary biogerontological discourse, however, presents a paradox because one of the distinctive claims of contemporary biogerontology, understood bio-molecular and the bio-demographic characterisation of the processes associated with chronologically older individuals, is that it provides an alternative model for understanding those diseases that are commonly associated with age, such as heart disease, stroke and cancer (Butler at al, 2008). Rather than pursuing the disease-specific model that has been deployed within many other branches of the biomedical enterprise, biogerontologists argue that increases in health and healthy life expectancy are more likely to be achieved by focusing research on the common biological basis of all those diseases that would seem to characterise the lives of the older people. In reconstructing these diseases as part of a wider set of 'degenerative diseases' that are only connected contingently to the

organism's chronological age, biogerontology would appear to call into question the status of ageing as a biologically distinct and biologically meaningful phenomenon. In other words, the development of biogerontology seems to undermine the possibility of any conjunction of biology and medicine which the phrase 'biomedicalization of aging' conjures almost by definition.

In this, we follow closely Keating and Cambrosio's (2003) definition of biomedicine as a new set of relationships and practices established in the second half of the 20th century which are concerned with the mediation between research laboratories and clinics through hybrid *bio-clinical* entities. Rather than a systematic application of biological standards and products to clinical work, biomedicine is structured around conventions that enable the difficult coordination between bench and bedside within specific diseases. In this paper, we argue that the paradox that biogerontology presents is intimately linked to a critique of biomedicine as it took shape during the second half of the twentieth century. We propose to articulate this understanding of biogerontology by first exploring the political and technical aspects of recent proposals that have emerged from within biogerontology to reorganise not just gerontological research, but biomedical research more generally. We then offer a genealogy of this situation by turning to the middle decades of the twentieth century, when gerontology first gained institutional recognition and the biology of ageing was a domain contested between those who argued for a 'basic' approach and those who proposed to focus on the differences between 'normal' and 'pathological' ageing. We then consider how the latter approach came to dominate the organisation of gerontological research in both the United Kingdom and the United States, but for very different reasons, and how it eventually was embedded in the activities of the National Institute of Aging (NIA). We then trace how the NIA's blue-print for research into the causes and treatment of ageing paradoxically initiated a transatlantic, biogerontological critique of biomedicine. In so doing, our aim is to establish that this critique is shaped by two interrelated and historically contingent conditions, namely the status of biogerontologists as 'outsiders' to the institutions of biomedicine, and, more importantly, the hybrid character of biomedicine itself. The development of biogerontology, we maintain, contrasts sharply with the many successful relationships that have been established between the laboratory and the clinic by means of mediating, hybrid versions of the 'normal' and the 'pathological'. If, as Boltanski and Thévenot argue, 'the less pure a situation is (in the sense that it contains objects from different worlds), the easier it is to denounce it' (1999: 374), biogerontology has harnessed the internal tensions of biomedicine, seeking to construct a field of action whereby the historical opposition of biomedicine and

public health no longer obtains, and the laboratory, preventative medicine and health maintenance programmes are instead integrated seamlessly (Rose, 2007). In the conclusion, we point however towards sources of uncertainty within the biogerontogical project that should be taken into account in further research.

Biogerontology and biomedicine

In past decade, gerontological research has gained public and policy recognition. In the United States, for example, a group of biogerontologists and policy makers who were attending the White House Conference on Aging (2005) have drawn attention to the minute proportion within the annual budgetary allocation for the National Institutes of Health that is dedicated to understanding the basic biology of ageing and called on Congress to reconsider its position because 'the aging research field [is] on the threshold of a new way of thinking – shifting from a focus on specific age related illnesses to a search for an understanding of aging itself (Alliance for Aging Research, 2005: 4). In the United Kingdom, the Science and Technology Committee of the House of Lords, following the advice from the leading British biogerontologist Thomas Kirkwood, has advanced a similar argument, noting that 'most of the research on ageing and health ... is focused on specific diseases and medical conditions for which age is the single largest risk factor' and then bemoaning the paucity of support for much more promising programmes of research on the 'basic processes of ageing' (House of Lords, 2005: 103).

The focal point of this critique is the dependence of existing approaches on clinical definitions of those diseases most commonly associated with old age. According to biogerontologists such as Kirkwood and the other, trans-Atlantic signatories to a position statement on health promotion and disease prevention in the twenty-first century (Butler et al., 2008), such dependence is troubling because it is an effect of an anatomical division of the body that was forged in the nineteenth century and no longer provides a useful way to understand disease. The clinical worldview, these biogerontologists maintain more specifically, was well-suited to pathologies characterised by discrete and specific aetiologies, but is inadequate to address the chronic, long-term illnesses of the late twentieth and early twenty-first centuries. The protracted temporal unfolding of these illnesses is so nearly coterminous with ageing that it unsettles the epistemic pairing of the 'normal' and the 'pathological' that underpins clinical perspective on ageing. Furthermore, this pairing assumes that the two states can be situated proximally and intervened upon directly, but this obscures understanding the diverse and complex processes involved in the declining

functional capacities of the organism such that the perspectives of the laboratory and clinic must be integrated with programmes of health screening and maintenance. A further criticism of biomedical research is that, by relying on methodological and epistemological structures that are wholly incommensurable with the phenomenon studied, it cannot but fail to deliver the treatments of those pathological states commonly associated with old age. As the House of Lords Science and Technology Committee (2006a) notes, 'generic research into the process of ageing ... may be 'the most direct route to developing novel interventions and therapies". In sum, the charge is that current organisation of biomedicine may serve many clinicians and biomedical researchers well, but it fails to secure health and longevity.

At the same time, the biogerontological critique of biomedicine also aims to redraw the contours of the relationship between the biology, medicine and society. As the following extract from an interview with a biogerontologist[i] intimates, what is required is a wholly new approach to disease. Having been asked about his expertise, this biogerontologist foregrounds the issue of normal ageing:

> [My] scientific interest is to explain the occurrence of age associated diseases. Some people call that ageing, some people call that normal ageing, [and] some people say it's different from normal ageing. I don't make a distinction between them.

When pressed on the importance attached today to research on the causes of Alzheimer's Disease as answer to the problems posed by old age, this biogerontologist adds:

> What's behind, let's say the senile muscle, is of equal importance ... because people can't go out any more and they suffer from it. What's normal? There's nothing like normal ageing.

What is important, according to this biogerontologist, is a historical disjunction between the genome and the environment to which it is exposed:

> [We] were not meant to live longer than forty years, and the system is optimised in an environment like Africa. But now we don't live in Africa. ... [Our] life history is ... the result of ... an old, optimised genome ... now ... exposed under modern, affluent conditions. But it's not meant to be ... it's not meant to be exposed under these conditions.

From this perspective, normality is a historically specific discrimination of little biological significance, at least insofar as biology is equated with attention to the evolutionary dynamics shaping the life course of the organism. This argument has gained considerable momentum in the context of what has become known as 'evolutionary medicine' (Nesse & Williams, 1996)

and encapsulates an approach to illness as natural phenomena that undermines recourse to categories of normal and pathological. The perspective of biogerontologists as exemplified in this interview is that the category of normal ageing is an arbitrary, historically contingent label. Through this category, problems that 'are of equal importance' are hierarchised focusing attention on 'illnesses' to the detriment of frailty as an effect of the organisation of medical specialities and illness markets they sustain.

How to understand this wholesome critique of biomedicine? Our proposal is that it is contextually rooted in biogerontologists' outsider status to the main enterprise of biomedicine as it took shape in the latter half of the 20th century. Because our thesis goes against the grain of most social analysis of the science of ageing, below we trace the complex process through which biogerontology achieved this position. We document the multiple attempts to forge a relationship between the biology of ageing, medicine and society from the 1950s onwards and how those were ultimately incorporated in biomedicine through an emphasis on the pathologies of old age rather than on ageing as a biological phenomenon. Given the aims of our paper, we are less interested in providing insights into the origins of research into the biology of ageing (Achenbaum, 1995; Park, 2008) and are mostly concerned with understanding how, at a particular historical juncture, when the biology of ageing became a topic of interest to various philanthropic institutions and the State, different epistemic projects were articulated and transformed.

Between unity of causes and unity of effects

Between the late 1930s and early 1950s, a variety of public and private actors became interested in the biology of ageing. Underpinned by actuarial projections of an 'ageing population' and established views that associated older people, economic deprivation and physical and mental illness (Haber and Gratton, 1994; Thane, 2000), such concerns however did not encounter a well formed and agreed set of epistemic commitments but instead a diversity of approaches were being formulated in controversies surrounding the definition of ageing as an object of research. Such divergence was particularly evident during the conferences supported by two of the most active philanthropic supporters of research on ageing, the Josiah Macy Foundation in the United States and the Nuffield Foundation in the United Kingdom.

The Josiah Macy Foundation was responsible for funding surveys of ageing in the 1930s and commissioning a seminal conference on the biology of ageing at the end of that decade, the Woods Hole Conference on the Problems of Aging (1937). As Park (2008) has documented,

Edmund Cowdry's edition of the published proceedings of this conference was ridden by disagreement between the various contributors, mostly around the 'parameters' and 'standards' by which to contrast normal and pathological ageing. Similarly, the Nuffield Foundation was a key sponsor of ageing research and is renowned for having sponsored the Survey Committee on the Problems of Ageing and the Care of People (1947), which brought the living conditions of older citizens to public attention (Thane, 2000). What is less known is Nuffield Foundation's support for research into the biology of ageing, which started in 1940, with Lord Nuffield personal interest in, and support for, Vladimir Korenchevski's research on the endocrinology of ageing, and expanded throughout the 1940s and 1950s into an impressive network of laboratories across British universities. Here too, however, the uncertainties were deep and, when the Nuffield Foundation sponsored a symposium in 1956 to showcase its achievements in this domain, there was conflict between those who wanted to find standards by which to contrast normal and pathological ageing and those who considered this endeavour as pointless (Yapp & Bourne, 1957).

The questions of definitions raised during these diverse encounters were most clearly delineated during the first CIBA Foundation Colloquium on Ageing, which was held in 1954 in London immediately after the first meeting of the International Gerontological Association. The list of participants included the leading experts of the day on the biology of ageing, ranging from Edmund Cowdry himself, his close associate, the experimental biologist Albert Lansing, Nathan Shock, the Director of the Gerontology Research Centre and founder of the International Gerontological Association, Peter Medawar, Professor of Zoology at University College London and his own close associate, Alex Comfort, Nuffield Research Fellow in the same department. According to the published proceedings of the colloquium, following R. E. Tunbridge's introduction of the concluding, general discussion, Comfort intervened in the following terms:

> I would like to put in a plea for [Professor Medawar's] definition of senescence as the increase in liability to die with advancing age. It may be proper to distinguish ageing from senescence, but in that case I think we can scrap ageing altogether and call it development, because gerontology is an entity which only comes into existence to describe a process human beings don't like, a deteriorative process, and I take it that it is senescence with which we are concerned here.
>
> Earlier in the meeting Dr. Lansing made a declaration of faith on the subject of the overall unity of the senescent process. I don't want to speak out of turn, but I'm somewhat sceptical of [the] underlying unity of any ageing process.

Comfort's call for a conventional definition of 'ageing' did not go unchallenged:

Lansing: But take the male rotifer: it is born, it has no alimentary trait and dies of starvation within twenty-four hours after fertilizing. Does he die of senescence? I'd rather put him in a special category, as a very degenerate character who starves to death in the twenty-four-hour period that he is busy fertilizing. [...] When I think of senescence I think of something that happens not to children or to infant rotifers, but to the organism that has become an adult and then undergone some type of change, to wind up dead sooner or later. That's what I mean by senescence. The maturation of the embryo, the new born child, the adolescent, the changes with time prior to maturation, to me are not senescence.

Cowdry: Yours is the downswing of life, then.

Lansing: Yes, after adulthood has been reached. I can't define adulthood too well, and in some cases the changes that occur in adulthood are said to be improvements rather than losses.

Cowdry: You don't have to define it if you just call it the downswing, that implies that after a height you start to go down.

Comfort: Do you agree then that for various organisms the factors that contribute to that downswing tend to differ very radically from phylum to phylum?

Lansing: I'm not prepared to agree to that. I think we have special cases which bring about death, but not all death is due to senescence. [...] The declaration of faith I made yesterday stems in part from the various types of survival curves that Dr. Comfort showed us. [...] It would be quite a coincidence if all these processes all expressed themselves in the same way.

Comfort: Raymond Pearl plotted a survival curve for automobiles which was again the same shape!

Shock: I think the argument that because two different phenomena can be made to fit the same mathematical formulation they have common processes behind them is an extremely hazardous one.

Lansing: I said only that it's a possibility, I'm not prepared to say that we have as many kinds of protoplasm as we have species. I think there is a common protoplasm with basic properties of multiplication and growth, decline, irritability and so on, varying in detail, not in principle.

Shock sought to bring the argument to a close in the following terms:

I would agree that protoplasm is probably fundamentally much the same stuff, although we know that various tissues develop different functions, so that their enzyme systems must vary quite widely between different cells in the same animal. To that extent, I would agree that perhaps if you knew what it was that caused a cell to lose its ability to maintain concentration gradients, maintain its metabolic processes, you would be a long way toward understanding the ageing process. But it seems to me that the techniques that we have for investigating single cells are very meagre. Dr. Cowdry feels that if you take a cell out of its tissue it is no longer a cell. If we accept this position we are limited to unicellular

> organisms for study, but unfortunately most of these species simply divide and form two new cells so that 'ageing' fails to occur. Thus, we are faced with the problem of studying more complex animals or tissue, using both biochemical and physiological techniques. Since changes in the environment of the cell, produced by changing the diet of the animal, will often result in alterations in cellular enzymes, it seems to me that perhaps we are going to have to look at the problem of ageing from a number of different levels simultaneously and not try at the moment to conceptualize the entire problem in one framework. Prof. Medawar has approached the problem from a statistical evaluation of life tables; I am not prepared to accept this approach as the only way out of the difficulties. I think the examination of life table might be an index as to what you were doing to a process, but if you are going to explain ageing as a process I think ultimately you have to look at individuals, and perhaps the best way is to look at them from different points of view and at different levels of organization. I doubt if it would be possible to formulate a definition of ageing that would be acceptable to everybody and would cover all the aspects of the problem as it now stands (Wolstenholme & Cameron, 1955: 240-244).

This debate can be regarded as the confrontation of three different visions for the biology of ageing, underpinned by disparate epistemic and political commitments. The first of these was that advanced by Medawar and Comfort. Theirs is the perspective of population geneticists working with life tables and relying on evolutionary arguments to explain differences between populations and species. On this account, ageing or senescence was an age-specific aggregation of biological phenomena that were only visible in 'domesticated animals and that were physiologically unrelated. This suggested that any interventions should operate at the aggregate, population level. Against this perspective, was that of experimental biologists such as Cowdry and Lansing, who relied on particular organisms to produce 'models' of physiological phenomena that were assumed to obtain across different species. From this perspective, ageing was to be regarded as a unitary phenomenon that occurred in all organisms at some point in their developmental cycle. Any interventions should operate at the cellular level. The third perspective, represented by Shock, was firstly that the first two perspectives were epistemologically equivalent, and, secondly, that the individual should be regarded as the fundamental biological unit which could then be examined 'from different points of view'. This programme was to be delivered by physiological measurements of the ageing individual in the laboratory, clinic or community so as to establish standards of 'normal ageing' and would leave to clinicians and physiologists the task of managing the pathologies of ageing.

Shock's ability to subsume the evolutionary and physiological perspectives was not underpinned by some alternative disciplinary approach. What allowed Shock to speak in such terms and with sufficient authority to close the debate, at least temporarily, were changes

happening elsewhere. His proposal to embrace 'different points of view' was predicated upon taking the individual as the unit of analysis in gerontological research because this vision was aligned with contextual shifts in American funding of medical research at that moment. His aim was to integrate gerontology in the institutional transformation sparked by the creation of the National Institutes of Health in 1946 and correlated private and public investments in research programmes on cancer and heart disease that have come to define the biomedical enterprise (see Gaudillière, 2002). It is to the project of integrating gerontology in biomedicine in the following decades that we now turn.

Coordinating medicine, biology and old age

There can be little doubt that, during the 1950s and 1960s, Nathan Shock played a pivotal role in integrating gerontological research within the changing institutional organisation of American medical research, and in aligning gerontology with the normative requirements of biomedical research. This was encapsulated primarily in the design of the Baltimore Longitudinal Study of Aging, which Shock began to develop in the 1950s to measure individual functional capacity over time and so establish normative definitions of ageing. Although contemporary with the longitudinal studies which characterised American and British public health research during these years, such as the Framingham Heart Study (see Oppenheimer, 2005; Rothstein, 2003), the Baltimore Longitudinal Study of Aging was distinctive insofar as it focussed on 'healthy individuals' alone, to the exclusion of all those who contracted any illnesses. This was due to Shock's interest in disentangling 'pure ageing' from 'disease' and so providing the standard that would guide geriatricians in their diagnosis and management of old age illnesses. This was in concert with the aims of geriatricians themselves.

From the 1940s onward, American geriatrics aimed to establish itself within medical specialities. As Hirshbein (2000) has suggested, this was achieved by evoking a notion of normal ageing and then defining the expertise of the geriatrician as dealing with the prevention and treatment of diseases of old age. Gerontology, particularly the physiological and functional measurement provided by case-controlled or longitudinal studies such as those that Shock proposed, was construed as providing the required biological standards. Shock's distinction between normal and pathological ageing served clinical but also political goals: when the plan to establish a national programme of research into the causes of the diseases commonly associated with old age finally moved onto the national political agenda in 1968, it

was predicated on this distinction between normal and pathological ageing (Achembaum, 1995).

However, while the distinction would appear to enable a hybrid understanding of normal ageing that was both workable in the clinic and the laboratory, gerontology remained relatively constrained by its institutional association with the Veterans Administration (Haber, 1986) such that it was only when large numbers of veterans started developing the diseases of old age that the calls to establish a national programme of research into the causes old age disease gained any support. In other words, the solidity of the alignment between Shock and the future National Institute of Ageing (NIA) should not be over-estimated and it perhaps is no surprise that, despite Shock's ambitions to create a national programme of research that might help to differentiate healthy from unhealthy ageing, the NIA was only fully recognised within the national funding institutions when it began consistently to sponsor research on what has today become the defining disease of old age, Alzheimer's Disease (Anon, 2008).

In the United Kingdom, a different configuration of medicine, biology and old age not only distanced gerontology from biology altogether, but resulted ultimately in the significant weakening of gerontology. Some of the reasons for this fate are most evident by comparing the disciplinary affiliations of the participants in the CIBA Foundation Colloquium and the Nuffield Trust conference on the 'biology of ageing'. While no social scientists appear to have been invited to attend the CIBA Foundation Symposium, other than in an honorary role, the conference funded by the Nuffield Trust was organised by the Frederick Le Gros Clark and Norman Pirie, a ergonomist and a plant biochemist. While the two shared the interest in promoting scientific solutions to pressing social and political problems that characterised the polymathic and politically engaged members of what Werskey (1979) has labelled the 'visible college', it denotes key uncertainties about the role of biology in what was increasingly being characterised as the 'ageing society'. Such divergent disciplinary expertise was also evident among the speakers invited, some being zoologists and others being either clinicians, psychologists or economists. The challenge was then to establish how their expertise might be co-ordinated so as to address the social and political question posed by ageing.

This situation is not surprising given that the Nuffield Foundation was renowned firstly as the sponsor of the Survey Committee on the Problems of Ageing and the Care of People and that, in the absence of any substantial, structured funding by the Medical Research Council (MRC) for research on the biology of ageing, it supported a variety of academic programmes that

relied on equally varied methods, though focussing primarily on the importance of social and economic conditions to the definition of 'normal' ageing. Furthermore, as Martin (1995) has observed, it was not through the laboratory but 'through the technique of the survey, [that] doctors created a body of knowledge relating to the social, economic, and medical needs of the aged population in their own districts' (458). In the process, British geriatricians defined gerontology as the field, in Lord Amulree's definition, concerned with 'those elderly sick with social and economic problems' (460). Importantly, this construction positioned gerontology outside the hospital, the main research platform of British biomedicine during the second half of the twentieth century (see also Stewart, 2008).

This association between the old age and that peculiarly British disciplinary integration of social and medical science that went by the name of 'social medicine' was responsible for much uncertainty around the place of the elderly within the National Health Service (NHS). Under these circumstances, any funding for research on the medical problems posed by the elderly tended to be allocated to disease specific programmes within the MRC because there seemed to be nothing so biologically and clinically distinctive and remarkable about the patients' chronological age as to deserve the attention of a specialist. Consequently, in the 1960s and 1970s, when social medicine lost its precarious institutional support within both the MRC and NHS(Porter, 1997), British gerontology lost all residual disciplinary legitimacy. Despite the fact that, at the time of the 1954 CIBA Foundation Colloquium, the evolutionary perspective on ageing was a wholly British and very vibrant current, institutional and political factors worked together to progressively disconnect biological explanations of ageing from any public debates and programmes to address the 'problem of old age', such that by the 1970s Peter Medawar could declare British gerontological research as moribund (Medawar and Medawar, 1977)

In sum, if the 'problem of old age' emerged during the years between the late 1930s and early 1950s as a pressing political question and a variety of powerful institutions became interested in the biology of ageing, the successful alignment of the 'problem of ageing' with biology and medicine was a highly contingent affair.

The trouble with the National Institute of Aging

The establishment of the National Institute of Aging (NIA) was a protracted matter. Although gerontological research had been integrated into the programme of the National Institutes of Health (NIH) since the latter's creation, this had not satisfied either biomedical researchers or policy makers. The eventual integration of gerontology in the National Institute of Child

Health and Human Development was not satisfactory either and gerontologists spent most of the 1960s lobbying on behalf of a federal programme that could 'coordinate research on the biological origins of aging' (Lockett, 1983: 85). Importantly, the drafting of legislation from 1968 onward to create the desired federal programme or institute proved very controversial, including a presidential veto in 1972. While such controversy has been portrayed as a matter of divergence between gerontologists and the medical establishment, there is evidence of divergence among gerontologists themselves over the framing of a coordinated programme of research on the biological origins of ageing. While one proposal was embodied by Nathan Shock's influential vision of a programme dedicated to the definition of normal ageing explored above, another originated with an outsider, Leonard Hayflick.

Between 1963 and 1965, Hayflick, a cytologist working in the expansive domain of experimental oncology, challenged the notion that cell lines were potentially immortal by demonstrating that the number of replications cells could undergo was limited and that the limit was fixed by cellular mechanisms that were eventually located within the nucleus (Landecker, 2007). The challenge went unnoticed among oncologists because the notion that cell lines were potentially immortal was too solidly embedded in the material practices of experimental oncology, but it did not go unnoticed among gerontologists insofar as it offered scope to expand the domain of gerontology beyond the confines of organs, individuals and populations and a re-articulation of Cowdry's experimental biology approach to ageing (see above). This interest came to a head in 1973, when Hayflick received the Robert Kleemeier Award, which the Gerontological Society of America bestowed annually 'in recognition of outstanding research in the field of gerontology'. In his acceptance lecture, Hayflick (1974), while admitting feeling 'somewhat uncomfortable in accepting an award for work which at the outset was undertaken with the biology of aging farthest from [his] mind' (37), was quick to propose the following:

> What are the implications to gerontologists of the notions that are emerging from cytogerontology? I believe that there are several important implications. The first is that the primary causes of age changes can no longer be thought of as resulting from events occurring at the supracellular level, i.e., at cell hierarchies from the tissue level and greater. *The cell is where the gerontological action lies*. I believe therefore that purely descriptive studies done at the tissue, organ and whole animal level, as they pertain to the biology of aging, are less likely to yield important information on mechanism than studies done at the cell and molecular level (39; our italics).

In other words, according to Hayflick, investigations of ageing would be most productive when grounded in the methods of 'cytogerontology', the new field of research which Hayflick himself was busy trying to define and delineate. More importantly, however, Hayflick seemed intent on challenging Shock's programme for the development of gerontology firstly because Shock had justified his focus on individuals on the grounds that the cellular level concerned only a subset of the gerontologial phenomena, and secondly because the list of 'purely descriptive studies' of ageing presumably included the Baltimore Longitudinal Study of Aging. But there was more. If Shock's programme was underpinned by the need to distinguish between normal and pathological aging, on Hayflick's vision this would become a problematic endeavour:

> One is forced to conclude that if all disease related causes of death were to be resolved, then the aging processes would present some clear physical manifestations well in advance of death itself. The challenge, of course, is to separate disease-related changes from the basic biological changes that are a part of the aging process. Since fundamental aging processes most certainly contribute to or allow for the expression of pathology, then the two concepts may be so closely intertwined as to make any clear distinctions a futile exercise in semantics (43).

The question about the relationship between the normal and pathological Hayflick thus posed rested explicitly on Alex Comfort's well-established evolutionary explanation of ageing (see above; Moreira & Palladino, 2008). Natural selection, Comfort argued drawing on Medawar, operated most forcefully on those phases of the life cycle which were related to reproduction, so that the expression of any deleterious mutations in these phases would be targeted more strongly than their expression in post-reproductive phases. This, according to Comfort, led to an accumulation of deleterious genes whose expression occurred in the later phases of the life cycle, eventually resulting in the genetic determination of the post-reproductive weakening of the organism commonly named 'ageing'. From this evolutionary perspective, seeking to 'separate disease-related changes from the basic biological changes that are a part of the aging process' was questionable, to say the least. Significantly, just two years after these critical declarations, Hayflick was mentioned as a possible first director of the NIA, thus illustrating the persuasive force of Hayflick's criticism of Shock's programme, but the ambition to totally reconfigure the organisation of gerontological research may also have been the reason for its limited institutionalisation.

In effect, the first director of the NIA was neither Shock nor Hayflick, but Robert Butler, an old-age psychiatrist with links to the Democratic Party. One of the greatest challenges

confronting Butler upon his appointment was the lack of research capacity and limited public interest in gerontology. While Butler's earliest work on the psychology of ageing had betrayed a preference for Shock's vision of gerontology (see Butler, 1963), when the new director of the NIA suggested that 'research on aging has shifted from its exclusive disease orientation toward a more comprehensive investigation of the normal, physiological changes with age' (Butler, 1977: 8), the evocation of normality should be seen, given what was argued above, as more immediately related to the political need to re-articulate how Americans viewed older citizen's role in society, than to the needs of clinicians working with older people.

Despite Butler and others' attempts to enrol policy makers and funders in the gerontologial project, in the first years of its establishment, the NIA struggled to secure steady human and financial resources. This was only to change with the advent of what Butler himself called the 'health politics of anguish' (Fox, 1989: 82), a alliance of activists, clinicians and politicians who called public attention to the abandonment experienced by sufferers of senile dementia. The prioritisation of Alzheimer's disease within the NIA, particular though its extra-mural programme headed by Zaven Kachakaturian also embodied an alignment with emerging, competition-driven innovation policies, based on collaborations between universities and companies and ideals of 'rational' therapeutic development from bench to bedside (Moreira, 2009). With the support thus secured, the NIA experienced an influx of researchers from other areas of biomedical research, an influx also encouraged by 'aggressive recruitment', as one informant has put. This process helped to transform the NIA's role in the American polity but in the form of a disease-specific programme, that was to deploy the Institute's majority of resources (Ballenger, 2006). This caused dissatisfaction amongst biologists of ageing.

As the biogerontologist Richard Miller (2002) has noted with regard to this situation, and plaintively so, 'senators' and voters' parents [die] of specific diseases' and are less likely to fund a general, 'basic' programme of research on ageing. Alzheimer's Disease firmly established the position of the NIA within the political and clinical worlds, but only by emphasising illness and thus betraying that mixture of science and political advocacy which, according Moody (2000), has always characterised gerontology. In this, the NIA's programme of research in the 1980s and 1990s superseded both Shock's and Hayflick's visions of research on ageing and its future development. If gerontologists felt that an opportunity had been a missed, however, this situation also created the conditions for an unlikely alliance between programmes aiming to distinguish between normal and

pathological ageing, on the one hand, and investigations of ageing at the cellular and sub-cellular level, on the other hand.

Biogerontology and the promise of health

During the 1990s, there was an emergence of interest in the evolutionary and biomolecular understanding of ageing. Couched in the institutional alignment of evolutionary models and genetic research made popular years earlier by Richard Dawkins *Selfish Gene* (1976), the evolutionary biology of ageing promise new articulations of the problem of old age and how to address it. This is powerfully illustrated in Thomas Kirkwood's work from the 1980s onward. Kirkwood re-articulated Comfort's evolutionary explanation by combining molecular and demographic analyses to advance the notion that the organism should be understood as the product of a process involving the balancing energetic investments in the somatic body (Kirkwood, 1977). Those investments were aimed at the enhancing the chances of successful reproduction of the germinal line, which would have to be balanced against the energy cost of these investments to the continuity of this same line. On this evolutionary understanding of ageing, attention is directed toward the molecular mechanisms involved in the preservation of genomic integrity, or, as Kirkwood has put it, toward 'the evolved capacity of somatic cells to carry out effective maintenance and repair' (Kirkwood & Austad, 2000: 235).

Importantly, in this new gerontological vision, the business of biology thus becomes to enhance the ability of the individual to approximate the immortal germinal line, although immortality itself is irretrievably denied by the evolutionary history of the human species. The hope is that this redefinition will at least result in maximising the biological effectiveness of the individual up to the moment of death. In this, gerontology ceases to be a field of clinical specialisation concerned with the diseases of a distinct population, the elderly, as these diseases are re-articulated as unfolding temporally onto antecedent risk factors and bio-molecular pathways. Biomolecular and demographic pathways of aging individuals who might be 'at risk' of developing diseases such as Alzheimer's or cardio-vascular are traced backward, to the earliest possible genetic, molecular, behavioural or clinical manifestations with the aim to develop multiple preventative interventions. These diseases then become part of a wider set of 'degenerative diseases' that are only connected contingently to the organism's chronological age.

Within this configuration of gerontology, all of these degenerative diseases might be said to entail 'ageing', but in so expanding its domain of application the term 'ageing' no longer

identifies a distinct biological process of its own kind. Equally importantly, because the domain of gerontology thus defined is resistive to any precise delimitation, it can become an object of interest for all clinical practitioners involved in managing degenerative diseases, from the primary care practitioners controlling their patients' hypertension to the specialised clinicians required to train these practitioners in the assessment of the earliest symptoms of illness. Furthermore, gerontology also offers opportunities of development to a great variety of actors in the market for health care as investigation of the mechanisms involved in the onset of these degenerative diseases greatly expands opportunities for companies because the threshold of treatment moves ever backward to encompass a greater fraction of the population. This said, the investigation of these same mechanisms also offers opportunities to those providing the wherewithal and support to secure 'healthy lifestyles' from birth to death. In so doing, biogerontology promises a central expectation of private and public health care insurers, namely reducing the prevalence of degenerative diseases so as to reduce the aggregate cost of provision.

This redefinition of gerontology draws on evolutionary biology to allow explorations of organisms' life histories in relation to genes and environment, so enabling links between the laboratory, preventative medicine and health maintenance programmes. In so doing, gerontology is not alone. One of the key changes in the organisation of research, clinical practice and policy in the end of the 20th century was the shift from the 'problem of disease' to the 'problem of health'. The focus is not on restoring health but on maintaining it and preventing disease. This entails not only constructing an understanding of the biomolecular, individual and social dynamics that lead to illness, but also a reliance on preventative therapeutic strategies and health promotion programmes. These in turn are sustained by enhanced epidemiological surveillance (screening, etc.) that regulate access to therapies and programmes through the identification of risk factors or states and the use of implements to support individuals's re-organisation of their conduct in light of such 'risks' (Armstrong 1995;Clarke et al., 2003; May et al, 2006; Rose, 2007). It is also here that biogerontologists seek to differentiate themselves from anti-ageing researchers and practitioners (e.g. Olshanky et al, 2002). While the later argue for an interventionist approach to ageing as a natural process (Mykytyn, 2008), biogerontologists, such as the one quoted earlier in this paper, would suggest that there is nothing natural or normal about ageing, it being the result of 'domestication'. The plasticity of the human organism, and in particular how first death and then ageing have been significantly postponed during the last few centuries, is where biogerontologists find support to propose further public health measures. Thus, nn a recent

public statement, , a number of influential biogerontologists have argued that 'the exploration of the mechanisms by which ageing can be postponed in laboratory models will yield new models of preventive medicine and health maintenance for people throughout life, and the same research will also inform a deeper understanding of how established interventions, such as exercise and healthy nutrition, contribute to lifelong wellbeing' (Butler et al, 2008: 399). In this, they call upon individuals identified through screening programmes and characterised through a variety of molecular and demographic markers to produce and maintain their own health and bring about the 'end of age' (Kirkwood, 1999). Such promise of health can only be realised however if innovation and research policies provide the means to focus on the basic biology of ageing and abandon biomedicine and its disease-driven business.

Conclusion

During the past decade, discontent with the organisation of research into the causes and treatment age-associated diseases has motivated public debates in both the United States and the United Kingdom. In this context, a number of influential biogerontologists have offered an alternative to disease specific programmes which calls into question both biomedicine and the historical opposition between biomedicine and public health. Such proposals, as we have argued elsewhere, can be taken as evidence of a transformation of socio-political forms of management of individuals and populations, in which 'the individual of the 19th-century bio-political imaginary, a human body whose biological constitution was irremediably fixed at birth, is giving way to an understanding of the human body as an assembly of bio-molecular components that can be […] recombined so as to maximize the resultant unit's cultural, social and political productivity' (Moreira and Palladino, 2008: 21; Rose, 2007; Deleuze, 1988). However, whether *and how* this link between the laboratory, preventative medicine and health maintenance programmes will work in practice is a matter of empirical case studies of the development, mediation and use of emerging gerontological technologies. In this paper, we have argued that these studies should not be underpinned by a 'critical' appraisal of biogerontology as a 'biomedicalisation of ageing' because biogerontology positions itself outside the institutions of biomedicine. The challenge will thus be to understand how the current uncertainties of the biogerontological programme will play out in multiple social and political arenas.

Such uncertainties are evident for example in the reluctance with which policy makers have greeted some of the proposal advances by biogerontologists. We use the British case to illustrate some of the issues involved.

The House of Lords Science and Technology Committee was particularly surprised that it was not the Science Secretary who responded to its report on the scientific aspects of ageing, but the Minister for Work and Pensions, who argued that 'old age' had long been a major concern of the Government and that it had already invested very heavily in the improvement of health and social care, as well as pensions. Citing a memorandum by its chief scientific advisor and leading biogerontologist, Thomas Kirkwood, the Committee wrote:

> It is particularly disappointing that the Government seem to wish to 'pigeon-hole' ageing research, as if ageing were an isolated, discrete problem, and that research into ageing must necessarily compete with research into other areas. Thus the response reproduces the familiar mantra that 'given finite resources, there will always be a need to balance competing priorities for research'. As we sought to demonstrate in our Report—a point repeated by Professor Kirkwood in his written comments—ageing is a continuum, affecting all of us all the time. He also reiterates the point made in our Report, that generic research into the process of ageing, far from being in competition with research into specific conditions affecting older people, may be 'the most direct route to developing novel interventions and therapies'. There is no sign of such holistic thinking in the Government response (House of Lords, 2006a).

The Committee's contrast between 'specific conditions affecting older people' and the notion that 'ageing is a continuum, affecting all of us all the time' was informed by Kirkwood's more specific observation that, '[T]here are scientific connections between birth, early years, childhood and adolescence that have major impacts on health and quality of life in middle and old age. These need much greater attention ...' (House of Lords, 2006b). What might explain the Department of Work and Pensions rejection of the proposals the Science and Technology Committee is that it remains committed to the needs of a specific subset of the population, the chronologically aged and their distinctive social problems. This indicates that significant obstacles are presented to the biogerontological programme, which stem from different modes of governance and which suggest synchronous relationships between biopolitical and disciplinary formations in the present moment (Moreira and Palladino, 2005).

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